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Features

ON THE MEANING OF THE GLUCOSE TOLERANCE CURVE*

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I. Introduction

Following the administration of a standard amount of glucose to a normal individual the glucose tolerance curve is characterized by a) a normal initial blood sugar concentration, b) a rise to a maximum value, at a rate which depends upon the rate and method of administration, c) a gradual decrease to a value lower than the initial one (hypoglycemic phase), and d) a gradual return to normal. In the diabetic patient, the glucose tolerance curve is characterized by: a) a high initial blood sugar concentration, b) a rise to a higher maximum than that attained in the normal individual, c) a delayed decline toward the initial level, and d) the absence of the hypoglycemic phase. (Fig. 1 and 5). The hyperglycemia which results from the administration of glucose is due to the fact that the rate of administration is greater than the rate of removal from the blood stream. Diabetic hyperglycemia is also due to a disproportion between the rate of sugar production and that of sugar utilization.

In the case of the glucose tolerance curve, the amount of sugar available is greater than the amount of sugar utilized, whereas, in the case of diabetes, hyperglycemia can be due either to overproduction or to under utilization.

Sixty years after Mering and Minkowski (1) produced diabetes by removing the pancreas of a dog and 27 years after Banting and Best (2) isolated the first insulin, there is still a difference of opinion on which of these two mechanisms prevails in diabetes. Similarly there is no agreement regarding the significance of the hypoglycemic phase of the glucose tolerance curve. This phase is generally attributed to overcompensation for the preceding hyperglycemia, and could be accomplished equally well by an increase in insulin secretion and glucose utilization (3-7), or by an inhibition of the glucose production by the liver (8). According to the first view the absence of the hypoglycemic phase in the glucose tolerance curve of the diabetic is due to increased ability to utilize glucose, according to the second view it is due to glucose overproduction.

The solution of the problem is important for theoretical and practical reasons. If hyperglycemia stimulates the insular tissue and produces a continuous overactivity which may result in exhaustion, both diabetes mellitus (9, 10) and hyperinsulinism (11) must be controlled with "painstaking care" (9). The diet must be carefully planned, the amounts of carbohydrate and carbohydrate precursors must be carefully measured, and the dose of insulin carefully determined, insuring adequate utilization of glucose, normal blood sugar concentration, and freedom from glycosuria. This is a difficult task requiring a careful study of the case, a highly individualized therapeutic program and intelligent co-operation on the part of the patient.

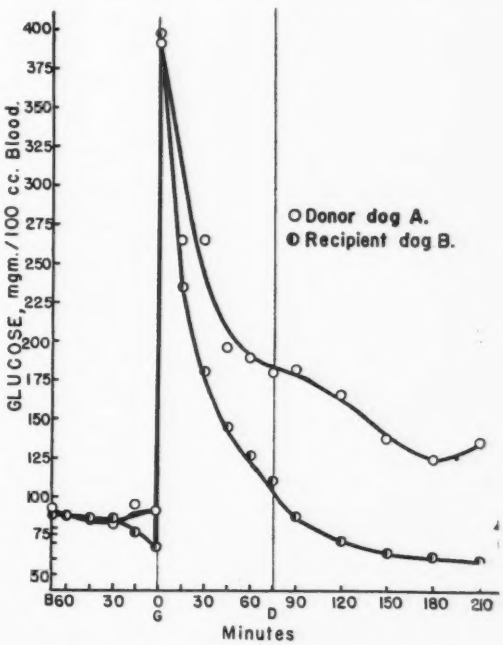

FIGURE 1. Pancreatic-femoral anastomosis. Normal dogs. Average of 3 experiments. B: before opening the anastomosis. G: intravenous injection of glucose into both dogs. D: anastomosis disconnected. Upper curve: donor dog A, lower curve: recipient dog B.

* Based on the Seminar of February 23, 1949.

On the other hand, if one believes that changes in the rate of liver glycogenolysis are the most important factor, the strict control of the blood sugar level becomes less important, the patient receives a more liberal diet, the amount of insulin limited to that which will insure optimal carbohydrate utilization and prevent ketosis, loss of weight and an excessive glycosuria with consequent loss of water and electrolytes.

The available evidence does not allow an unequivocable choice between the two points of view. v. Noorden (12) observed that pancreatectomy is followed by an increase in the amount of sugar in the blood of the hepatic veins and, on the basis of this and other observations, first suggested the theory of overproduction in diabetes. Bouckaert and deDuve (13) studied the amount of glucose required to maintain the blood sugar constant in dogs with and without liver and with and without pancreas and came to the conclusion that insulin has very little effect on the glucose consumption of skeletal muscle and that it acts primarily by promoting glucose uptake by the liver. These authors further suggested that the fate of glucose disappearing from the blood stream under the effect of insulin is not different from the fate of glucose disappearing under the mass-action effect of high blood sugar itself. Later, Soskin and Levine (14) confirmed and expanded these observations and reached the conclusion that the removal of the pancreas does not decrease the utilization of glucose by peripheral tissues and that a depancreatized animal consumes as much glucose at high blood sugar levels as does a non-depancreatized dog at normal levels. According to Soskin and Levine, the primary action of insulin is to determine the blood glucose concentration at which the glucose output of the liver starts and at which glucose utilization occurs. Thus in the presence of insulin the sugar output of the liver would be inhibited and sugar utilization possible at normal blood sugar concentration, whereas, in diabetes liver glycogenolysis would be unchecked, glucose overproduction would result in a rise of blood sugar and in glycosuria. The blood sugar concentration would continue to rise until it reached a value at which a normal utilization was possible despite the lack of insulin. Soskin and Levine further observed that the glucose tolerance curve of a depancreatized dog receiving a constant amount of insulin by continuous intravenous infusion shows the hypoglycemic phase, whereas the curve of a liverless dog does not. The authors conclude that the hypoglycemic phase cannot be due to stimulation of the islets of Langerhans, but must be due to the homeostatic regulation of liver glycogenolysis.

On the other hand, Crandall and Lipscomb (15) found, by direct measurement, that the hepatic glucose output does not change after pancreatectomy but the glucose consumption decreases from a normal of about 77 mg./kg./hr. to about 44 mg./kg./hr.

If the theory of overproduction is correct it must explain the origine of all glucose which is utilized, plus all the glucose which is excreted in the urine. When all the liver glycogen is exhausted, which may happen in severe diabetes, all the excreted glycose must be derived from non-carbohydrate sources by a process of gluconeogenesis. Gluconeogenesis from protein is associated with the urinary excretion of the nitrogen derived from the deaminized amino acids. In diabetes the amount of urinary nitrogen does not account for a sufficient amount of glucose. One must therefore, postulate the transformation of a certain amount of fatty acids into glucose. Recently Chaikoff and collaborators (19) found that this conversion although measurable with the isotope technique is not of great magnitude. Sevringhaus (5) studying Soskin's experimental records, came to the conclusion that the inhibition of liver glycogenolysis did not coincide in time with the hypoglycemic phase of the glucose tolerance curve and could not be the cause of it. More recently Bondy and collaborators (16, 17) catheterized the hepatic blood vessels of man and found that although liver glycogenolysis is inhibited by hyperglycemia, it returns to normal when the blood sugar has decreased to about 175 mg. per cent, which is not a hypoglycemic level. Waters and Best (18) express the opinion that the hypoglycemic phase is due to increased insulin secretion and point out that Soskin's depancreatized dog received by intravenous injection 3 to 4 times the amount of insulin required by a normal dog and could therefore mobilize a sufficient amount of it from their tissues to give a normal glucose tolerance curve despite the absence of the pancreas.

It may be seen from the above that the over-

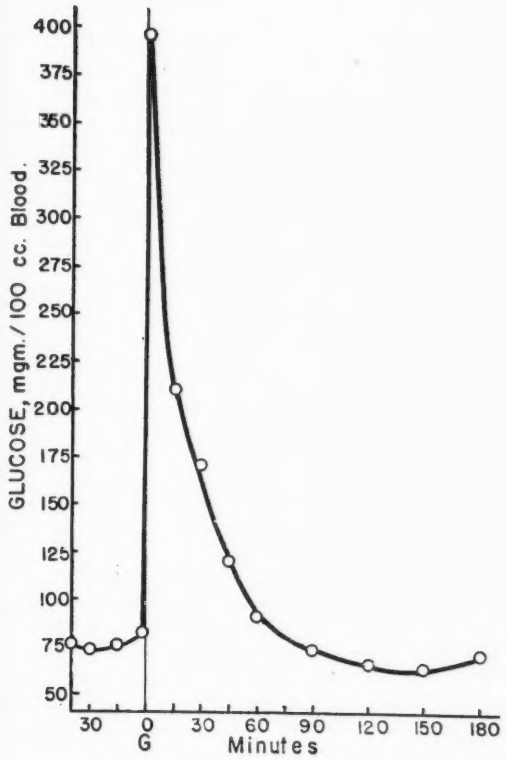

FIGURE 2. Glucose tolerance curve. Normal dogs. Average of 4 experiments. G as in figure 1.

production theory has been strongly advocated and sharply criticized. The same is true of the under-utilization theory, which has been thoroughly summarized and criticized (14). It will be sufficient to mention here some of the more recent pertinent observations:

(a) The glucose uptake by the diaphragm of the diabetic rat is depressed (20), and can be restored to normal by insulin (20, 21).
(b) Insulin stimulates the glucose uptake of the isolated muscle (22-26)
(c) Insulin increases the tolerance of eviscerated rats for intravenous glucose (27).

It would appear that glucose utilization is depressed in diabetes and increased by insulin.

Evidence is available that hyperglycemia stimulates insulin secretion. This hypothesis has been recently discussed and summarized (28). It is therefore possible that the hyperglycemia which follows glucose administration stimulates the pancreas. The resulting insulin secretion would cause hypoglycemia. Final proof requires a quantitative determination of the insulin output of the pancreas for which accurate methods are not availale. bThe need for such methods has been pointed out recently (18).

II. Experimental.

The effect of hyperglycemia on the insulin output was reinvestigated in this laboratory by means of cross-circulation experiments (28). A large dog was anesthetized with sodium amytal and its pancreato-duodenal and one of the femoral veins were cannulated. The other femoral vein was exposed. This was called the "donor" dog or dog A. A second, smaller dog was also anesthetized with sodium amytal. One femoral artery and one femoral vein were cannulated, the other femoral vein was exposed. This was called the "recipient" dog or dog B. Blood coagulation was prevented with heparin. The pancreaticoduodenal vein of Dog A was then anastomosed with one of the femoral veins of dog B, and the femoral artery of dog B was anastomosed with one of the femoral veins of dog A, with plastic tubing. In this manner the venous blood from the pancreas of dog A ran into dog B and an equal amount of blood returned from the general cir-

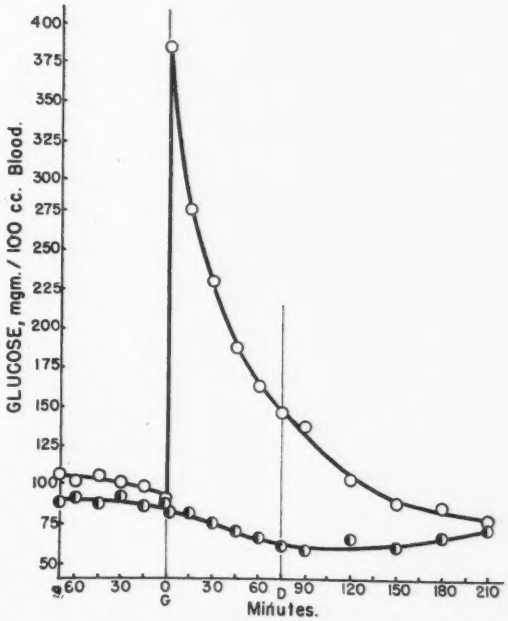

FIGURE 3. Pancreatic-femoral anastomosis. Normal dogs. Average of 5 experiments. G: intravenous injection of glucose into the donor dog. Other symbols as in figure 1.

culation of dog B into the general circulation of dog A, in order to keep the blood pressure of the two dogs at approximately their original levels. It was hoped that this technique would allow the detection of changes in the amount of insulin secreted by the pancreas of the donor dog by the changes in the blood sugar concentration of the recipient. Having opened the anastomoses, 4 blood samples were taken from each dog at 15 minute intervals to determine the basal blood sugar concentration. After this control period, 1 gram of glucose per kilo of body weight was injected simultaneously into both dogs. Figure 1 shows that the glucose tolerance curve thus obtained in dog B is identical with the normal glucose tolerance curve (figure 2) and shows a well-defined hypoglycemic phase. On the other hand, the glucose tolerance curve of dog A is prolonged and shows no hypoglycemic phase. We have interpreted these results to indicate that dog A is deprived of most of its insulin which is transferred to dog B through the anastomosis. If glucose is injected only into dog A (Figure 3) the blood sugar of the latter increases rapidly to a maximum while the blood sugar of dog B decreases to hypoglycemic levels and does not return to normal until after the anastomosis has been disconnected. This phenomenon suggests

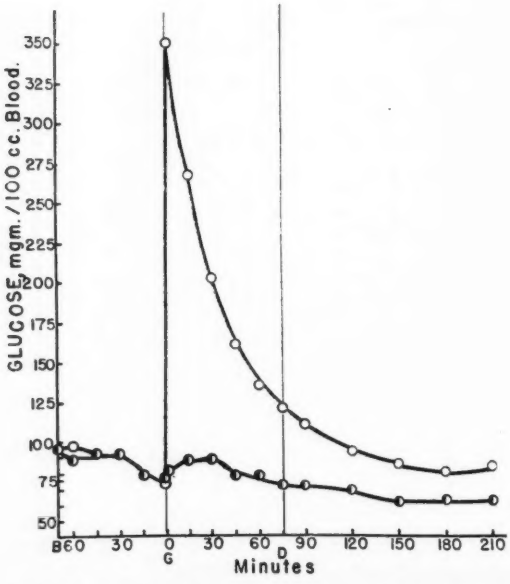

FIGURE 4. Mesenteric-femoral anastomosis. Normal dogs. Average of 6 experiments. Symbols as in figure 1.

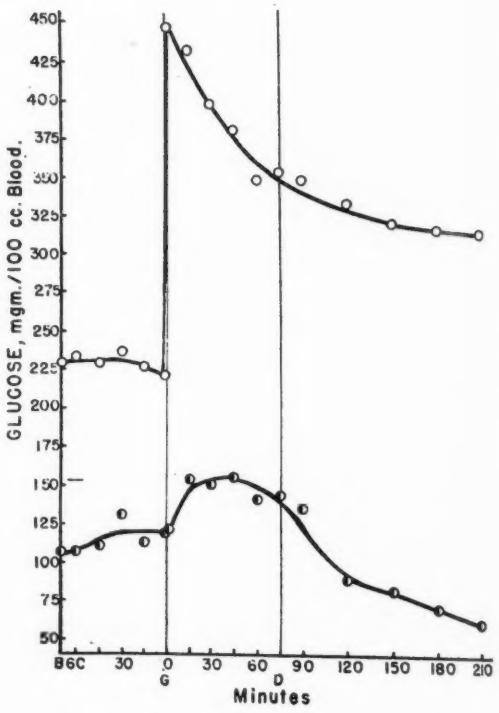

FIGURE 5. Pancreatic-femoral anastomosis. Alloxan diabetic donor. Normal recipient. Average of 3 experiments. Symbols as in figure 1.

that the hyperglycemia produced by the injection of glucose into dog A stimulates its pancreas to produce more insulin. This insulin is then carried by the anastomosis into dog B where it causes a fall in blood sugar. This conclusion is further supported by the fact that when the anastomosis was made between the mesenteric vein of dog A and the femoral vein of dog B (Figure 4), the hyperglycemia of A was not followed by a fall in the blood sugar of B, but rather by a slight rise. This rise is probably due to the glucose transferred from A to B and adds significance to the hypoglycemia observed in the recipient dogs when they receive pancreatic blood from their donors. Similarly, if the pancreatic blood is derived from an alloxan diabetic donor, which is unable to produce insulin, the injection of glucose into dog A (Figure 5) is followed by a rise in the blood sugar concentration of dog B. The injection of saline into dog A fails to produce the sharp decline in the blood sugar of dog B observed after the injection of glucose (figure 6).

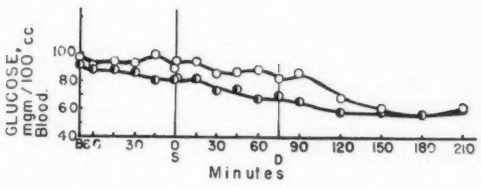

FIGURE 6. Pancreatic-femoral anastomosis. Normal dogs Average of 5 experiments. Symbols as in figure 1.

We believe that the hypoglycemia observed in the recipient dog following the injection of glucose into the donor is probably the result of a specific increase of insulin secretion by the pancreas of the latter brought about by the hyperglycemia. It will be noted (Figures 2 and 3) that the magnitude and the duration of the hypoglycemia of dog B are similar to those observed in the normal glucose tolerance curve. It appears likely, therefore, that in the intact normal animal the concentration of glucose in the blood regulates insulin secretion and by so doing it regulates itself. Although other mechanisms to secure constancy of the blood glucose concentration are undoubtedly available, the pancreas plays the primary role.

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THE EYES AND EARS IN MEDICAL EDUCATION

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It would be foolhardy for anyone to question the value of sight and hearing. Without both of these special senses the average human being would be almost totally helpless. Of course, there are rare exceptions where by means of a highly developed sense of touch very useful lives have been possible. Nevertheless, few would have the fortitude to surmount the overwhelming handicaps of one who is blind, totally deaf, and perhaps dumb.

Any one of the special senses, like one's good health, is not apt to be appreciated fully until it is lost. Needless to state, possessions of value are worth taking care of and should be used to the best advantage. No one is likely to deny these assertions. Nevertheless, normal eyes and

ears do not at all times accomplish the purposes for which they were intended. A student with normal hearing may sit through a lecture, but he does not listen. Consequently, his time is wasted, for he has failed to gain knowledge that other members of the class have acquired. Or the student may listen intently, but he fails to comprehend what he hears. In the latter event, the fault may be charged to the teacher, through lack of clarity. Again, it may transpire that the student misunderstood what the instructor said. Dr. Walter Stanley Haines, who was Professor of Chemistry at Rush Medical College for fifty years, once told me an interesting story. He stated it had been estimated that one-third of an audience grasped all that a lecturer said; one-third understood him to say the opposite, and a third really comprehended nothing. However, a possibility of that kind was not applicable to the students of Dr. Haines, who was one of the most ideal teachers that ever graced the faculty of a medical school.

Many times I have repeated an incident which illustrates the relative importance of the eyes and ears in the practice of medicine. An interne had just completed writing the history of a man in the receiving room of a large hospital. The attending physician chanced by, glanced at the patient who was lying on a cart nearby, and said, "What have you got?" The interne replied the patient was sent in as a case of pneumonia and handed the chart to the senior physician. "Good heavens!" said the latter, "what does this mean? You state in your history that the man lost his right leg in World War I. I just looked at him; he has had no amputation." "Well," said the interne rather indignantly, "I have put down in the history exactly what he told me."

The history of a patient's illness is a matter of importance. Past and present teaching has always placed great emphasis on this point. Nevertheless, when the physical examination follows the taking of the history, the physician involuntarily seeks to justify by his findings the complaints or circumstances which have been recited by the patient. Would it not be better if the student were taught to make routinely the physical examination first and then obtain the history? Uninfluenced by a history, the student should not be placed in the embarrassing position of describing conditions which are not present. Possibly the best organized and most complete history I have ever seen was written by an interne who described a disease that the patient did not have. As a rule, the primary purpose of a history should be to serve as an aid in confirming the physical findings. A diagnosis based on the history may be totally erroneous. If the history is untrue, it may lead to serious mistakes, respecting not only the patient but his family and friends as well.

Among the exanthemata, the history is inconsequential in so far as the diagnosis is concerned. A patient with a measles rash has measles, and one with a smallpox eruption has smallpox. There is no history that can change a correct diagnosis which has been established by the eyesight. However, one may argue that it is not sufficient to depend upon vision, because the rash's etiology may not be recognized by the observer. Such a criticism is justified and emphasizes the importance of clinical instruction. "What the book says" does not always convey the picture that the student anticipates.

To acquire a knowledge of diagnosis of the eruptive diseases, there is nothing so valuable to the student as clinical material. But to simply view the patients is not sufficient. It is necessary not merely to look, but also to see. Unfortunately, everyone does not see the things at which they look. Observation is the essential requirement for diagnosis of the exanthemata, and yet it is insufficient unless one has learned to recognize what the eye perceives. Consequently, it is apparent that under appropriate circumstances no other method of acquiring medical knowledge can be as successful as visual education. The distribution and character of an eruption will denote frequently not only the nature of the disease but also the duration of the illness. The patient's history may supply corroborative evidence, but it cannot change what the eye detects. Observation is the foundation on which to build a reliable diagnosis; whereas, a conclusion is often dubious when it rests on a supporting history that may prove to be weak.

Possibly I am prejudiced in considering the main subjects of this brief discussion, but it is my firm conviction that the eyes have it.

THE CHEMIST AS AN AID IN THE DETECTION OF CRIME

By WM. D. McNALLY, A.B., M.D.

Chicago, Illnois

THE deleterious effects of poisons including those due to the bite of venomous vipers were probably known to prehistoric man. The most ancient writings indicate a knowledge of poisons in the early civilizations of Egypt and India. The crime of secret poisoning was known to the ancient Greeks and Romans and to the people of the far East before them.

Vegetable poisons extracted from plants were used by the ancient poisoners more often than mineral poisons. Bichloride of mercury was known to the Arabian and Greek alchemists of the eleventh century. During the latter part of the seventeenth and the early part of the eighteenth centuries, the use of poisons as an agent of secret murder, became very common in Italy and France. La Spara in 1659 gave poisons to young women who wished to rid themselves of their husbands. At a later period, the notorious Toffana carried on a similar traffic at Naples. She poisoned nearly 600 people. The poison used by these women was arsenic. In France there was the Marchioness of Brinvilliers who poisoned her father and two brothers, and attempted to poison her sister with the aid of her accomplices. After confessing the crime she was tried and executed in 1676.

Women have figured prominently in the cold-blooded, calculating cruelty, and the wholesale character of their crimes.

Previous to the nineteenth century the methods for the detection of poisoning, as far as they were of value, depended largely upon the circumstances attending the administration. Apart from these they were superstitions, modified only to slight extent, even as late as the middle of the eighteenth century, by the more extended knowledge of the postmortem appearances in the cadaver caused by disease. The development of methods in medicolegal recognition of poisoning, to the point which they have reached at the present time, and which we believe far short of what they will be in the future, has followed the advances made in those sciences upon which they mainly depend, chemistry and pathology. Of the two forms of postmortem investigation of poisoning, toxicological chemistry in its more simple form was a well developed science half a century before the dawn of modern pathology. Inasmuch as the chemistry of the inorganic kingdom preceded the organic, the detection of mineral poisons became possible at an early date, while chemical proof of the presence of at least some of the vegetable and organic poisons is even at present unattainable by the means now at our disposal.

In the past several years the ultraviolet ray, the x-ray, and the spectrographic methods, have added a great deal to our knowledge concerning the presence of many substances which could not otherwise be detected by purely chemical means.

The Pathologist, after he has made a postmortem, is often unable to determine the cause of death without first having a chemical examination of the organs made. The material taken at autopsy is given to a chemist for analysis to make certain as to whether or not a poison caused the death, for in many cases of poisoning a characteristic appearance at postmortem is not found, and it is only after a chemical examination has been made that the poisoning can or cannot be excluded as a factor in causing the death of the individual. A careful history taken by a deputy coroner, will often give the chemist a clue as to what to look for thus enabling him to shorten the time of the analysis. In one instance the pathologist was unable to determine the cause of death and he submitted the organs to me for examination. A deputy coroner previously had given me the information that the deceased had taken a capsule at three o'clock in the afternoon and another at six o'clock and died one hour after the ingestion of the second capsule; he was also able to give the deceased's symptoms shortly before his death. These symptoms related could have been caused by one of two drugs, veratrum viride or aconite. Having in my possession the box with the capsules, I called up the druggist who filled the prescription and asked him to give me the ingredients which, he stated, in addition to a half dozen other drugs

contained one grain of aconitine. When I called his attention to the fact that he had a fatal dose in each capsule he became greatly alarmed because he had failed to notice that when filling the prescription. The physician who prescribed this dose claimed that it could be given safely and that he would submit certain authorities to substantiate this claim the following morning, but instead he came in and acknowledged his mistake. The average dose of aconitine is 1/400 of a grain, while each of these capsules contained twenty times that amount. This drug has been deleted in the U.S.P. XIII. The newspapers frequently mention the name of Caesar Borgia in connection with poisoning. This man who lived during the latter part of the fifteenth century, is credited with the poisoning of seven people. We have had instances in Chicago of individuals who caused more deaths by poisoning than did Borgia. In 1938, in Hungary, two women sold arsenic to other women who wanted to become rid of their husbands for their insurance. These women were caught and convicted. During the year 1939, a clique of murderers was uncovered in Pennsylvania, which allegedly had poisoned several dozen people.

In the history of toxicological chemistry, three events may be cited as marking important changes in the development of this science.

1. Practical application of the Marsh test in 1836 to detect very minute quantities of arsenic. This is still the standard test for arsenic. However, shorter ones such as the Reinsch, Gutzeit and Polarograph tests have been introduced.
2. The introduction of the Fresenius von Babo method of digesting organs for mineral poisons.
3. The separation of vegetable alkaloids. Since then a great improvement has been made in the methods of detection of poisons.

The proof, in a trial for murder, is divisible into three parts:

1. That the deceased died from the effects of poison.
2. That the said poison was administered to the deceased by the defendant.
3. That the administration of poison was deliberate and premeditated.

In a case investigated a man died after a three day's illness. A physician who attended him gave the cause of death as "sarcoma of the pylorus." A sister of the deceased came into the coroner's office and asked to have the body exhumed, as she feared foul play. A chemical examination of the organs taken at autopsy showed that death was due to arsenical poisoning. Upon investigation it was found that the wife of the deceased had been friendly with a former boarder who, at the death of her husband, again became a boarder in her home. When informed by a detective that her husband died of arsenical poisoning, she stated that her husband drank the liquid from a dish in which a poisonous fly-paper had been immersed. During her husband's illness, however, she did not inform the physicians who attended him of this occurrence. Love was the motive here.

Of all the poison cases of recent years none has attracted more attention than the Orpet case. This case appeared in the feature section of a recent Sunday Herald-American. Motive paralleled motive, action paralleled action, opportunity paralleled opportunity. The case had many points in favor of suicide and many in favor of murder. The instrument of death could have been the same in each case. Orpet, becoming infatuated with another girl, could have had love and passion as his motive. Each had access to cyanide, but the evidence indicated that Orpet had access to only sodium cyanide. Laying aside Orpet's own compromising conduct, his guilt or innocence rested entirely upon the chemical evidence introduced. Analysis of the stomach contents of the deceased girl showed that death was due to potassium cyanide. Analysis of samples of cyanide from the Orpet greenhouse, showed it to be sodium cyanide. Several months after the finding of Marion Lambert's body in Helm's woods, her coat was submitted to a chemist. On June 13, I was asked to examine the coat and other apparel worn by her. On the coat, about fifteen inches from the button hole, were three spots about one-half inch in diameter and one-quarter of an inch apart in a straight line. Only by a miracle could these spots have been arranged in such a manner. In the examination of hundreds of blood stains and other spots found on clothing, I have never observed a parallel case. While examining the spots, I noticed an odor of cyanide to which I called the attention of the State's expert, but the importance of this observation

was not realized until about a week to ten days later. Experiments with solid potassium and solid sodium cyanide on clothing and chemical glassware, failed to duplicate the spots, the resulting inference being that if the spots on the coat were ligitimate, they must have been made by a liquid recently—and to this fact I testified, because further experiments also developed the fact that solutions of cyanide when deposited on clothing, and glassware, lose their characteristic odor of cyanide in a short time. Experimenting with solutions of sodium and potassium cyanide, I was unable to duplicate the spots found on the coat, as a saturated solution of cyanide rolled off the fabric like a drop of mercury. The above facts were submitted to the State's Attorney of Lake County who was told that the same information would be given to the attorneys for the defense. Upon receiving this information the defense immediately claimed that the spots had been planted. If the coat had been photographed when first found, the contention of the "plant" could have been easily avoided. After the acquittal of Orpet, the coat with the spots was submitted by the Evening American paper to two chemists who found that the spots were made by sodium cyanide, an entirely different substance from that which I found in the stomach of the deceased girl. Great stress had been laid by the defense upon the findings of the chemical examination and that was, I believe, the main point upon which the jury acquitted Orpet.

The Tillie Klimek case was of special interest to me because the detection of this famous poisoning case came directly from a diagnosis which I had made. A West side physician had read in the papers of the many deaths in Cook County caused by alcoholism and he noticed the frequent mention of my name in connection with these cases. With this in mind, he reported to me that he had a peculiar case of alcoholism about which he would like to consult with me. After examining his patient I gave him my opinion that the man was suffering from arsenical poisoning. Chemical examination of this man's execretions gave positive proof of arsenic. It was then found that Tillie Klimek, the victim's wife, had had several husbands who had died under suspicious circumstances. When arrested and questioned, she admitted she had administered a powder to her husband given her by a cousin. Twelve bodies exhumed, including those of some relatives, gave evidence of arsenical poisoning. To two of her husbands who had taken ill, Mrs. Klimek brought in her mourning apparel to show them how she would be attired after their death. She was given a life sentence, while her cousin was acquitted.

Twenty-five years ago when I wanted to find out how much alcohol was in the organs or blood, I would use 100 grams of sample. Today we use only 0.1 c.c. of blood. In 1944, Dr. Harold Coleman and myself reported a method where four samples could be run simultaneously using 0.1 c.c. of blood.

With the rise in the consumption of alcohol a similar rise occurs in the number of murders. I reported this in 1924 in the Journal of the American Medical Association showing the effect of alcohol before prohibition and during prohibition. Blood alcohol determinations should be run upon every murder case.

The importance of having articles submitted for the presence of blood in cases of homicide justified itself so many times in my experience while chemist to the Coroner of Cook County (Illinois), that I believe it should be a routine procedure for Coroners and police officials to collect and submit all substances that contain suspicious stains to the chemist for examination. Although clothing may not contain bloodstains, it may contain dust, sawdust, or fibers which would connect the individual with the alleged crime. Anything that appears at all suspicious should be taken, such as: articles of clothing, bedding, pieces of wallpaper, curtains, stains found on woodwork or floors, or other substances containing suspicious stains found in the vicinity of the deceased, on the person of the accused or suspected, and on any implements, or weapons that may possibly have any connection with the case. Photographs should always be taken of the stains.

After all suspected substances are in the possession of the expert they should be kept under lock and key. This applies also to pathological material submitted for analysis, one man should be held responsible for their security. The most important stains submitted for medico-legal examination are: (1) bloodstains; (2) stains containing blood, such as: menstrual, lochial, rape, adultery, or sodomy; (4) stains containing mucus

or pus, such as: nasal, leukorrheal, and gonorrheal.

In the Peter LaLonde case, Saulte St. Marie, Michigan, January, 1914, expert testimony was the main factor in obtaining the maximum sentence of life imprisonment. A groceryman had been shot with a shotgun while stooping over taking pork out of a barrel. His money, amounting to several hundred dollars, was missing. Many suspects were picked up by the police. Suspicion was directed to a halfbreed Indian named LaLonde, who had been recently spending money very freely. When questioned he claimed he had obtained the money from the sale of an interest in a mine, the subsequent investigation of which proved it to be false. Several bills of paper money in his possession had bloodstains on it. His clothing had numerous spots of blood spattered upon it, which he claimed were due to blood from fish which he had been cleaning. A two-dollar bill, his clothing, shoes and the shotgun were submitted to me for examination. The stains were found to be made by human blood. Conviction followed.

After Loeb and Leopold were arrested, in the Bobby Frank case, I examined the clothing and boots of both suspects ,and also the automobile in which they took him to his place of concealment in the forest preserve. It was here that a pair of eye-glasses were found, which by clever detective work were traced to one of the boys. On the clothing and the automobile I found traces of blood of human origin. On the chisel wound with tape with which they had struck the boy on the head, I also found stains of human blood. The chisel had been thrown from the car in which they were taking the boy and someone picked it up; it was traced to a hardware store from which one of the boys had previously purchased it.

We are frequently asked to type the blood of a murdered person against blood stains found upon the clothing of a suspect. This is not satisfactory because, the blood may be hemolized and we are not able to obtain cells or even serum from the clothing. In a small percentage of cases we have been able to determine the group. In one instance I recall the case of a small boy who was walking along on the highway a few feet away from the pavement when an automobile coming at high speed, jumped the curb, struck the boy and continued on its way. The car subsequently located by the police was found to belong to a salesman of a packing company. A broken handle from the left door of the car was submitted to me for examination. An attempt had been made to wipe all the blood from the car, but it proved rather unsuccessful because some of the blood remained in the grooves of the handle, the examination of which showed that it belonged to Group II. The boy and his father were asked to come to my office for a blood examination and the boy was found to belong under Group II.

It is impossible to tell the exact age of a bloodstain, as so many factors enter into the drying of blood. Human blood may be detected in clothing and weapons after a period of many years. Blood has been deteced on the bodies of mummies buried a thousand or more years.

By the precipitin test used in the foregoing blood tests, we are able to show that the blood serum of man is closely related to the blood sera of apes, the steps of relationship from man backwards being definitely traceable. This we dislike to admit, but the Darwinian theory that our ancestors belonged to the higher apes is greatly strengthened by the precipitin reaction in showing blood relationship. Zoologists are in agreement today in placing men and apes in one order, the Anthropoides.

Most of the cases of death that I have investigated while toxicologist for the Coroner's office of Cook County, were those where there was a suspicion of foul play or where the coroner's physician was unable to determine the cause of death.

When organs are submitted to the toxicologist, each organ is weighed and described; any peculiar odor is noted and the organs are ground up separately by means of a meat grinder similar to that used by your mother or wife when preparing hamburger or meat loaf. The sample is thoroughly mixed, and an aliquot part is taken for the examination for arsenic, mercury and antimony.

As a preliminary test for the presence of these metals, we use a very simple method known as the Reinsch test. The material is heated in a casserole with hydrocholoric acid water, and a little strip of copper (the copper and hydrochloric acid having been previously tested for the presence of arsenic and mercury). If mercury is present the foil will have a silvery appearance due to the

deposition of the mercury. If the mercury is present in very small amount, this appearance will not be noted. If arsenic is present the foil becomes steel grey to black. The foil is washed with water, alcohol, and ether, and placed in the long arm of the subliming tube, which is nothing more than a piece of glass tubing with a constricted area of about one inch long. This constricted portion is refrigerated by a piece of filter paper dipped in cold water; heat is applied to the long arm of this tube with the finger or thumb closing the opening at that end. In the presence of mercury, shiny, silvery globules will be seen when the constricted area is examined under the microscope. If arsenic was present in the original material, octahedral crystals of arsenic trioxide will be desposited in the constricted portion and can be seen under the microscope. For the quantitative examination of arsenic, we make use of the Marsh and the Gutzeit tests. For the examination of other metals we use the Fresenius von Babo method, digesting the tissues with hydrochloric acid and sodium chlorate. Special methods are used for the isolation of a number of these metals.

In the early days lead in urine or organs was determined by the sulphate method, later by the chromic acid, dithizone and still later by the dropping mercury electrode or Polarographic method which is quicker and more accurate. For the examination of volatile poisons we use an apparatus (such as is shown in Slide No. 4, which consists of a copper boiler used as a steam generator, connected to a Kjeldahl flask in which is placed an aliquot part of the tissues. This in turn is connected to a Leibig condenser with an adapter which dips into a small amount of distilled water in an Erlenmeyer flask, which is used to collect the distillate. In the distillate will be found grain alcohol, wood alcohol, cyanide, carbolic acid, chloroform and other volatile substances distilling with steam from acid solution. The contents of the Kjeldahl flask are made alkaline usually with magnesium oxide, which releases substances that distill from alkaline solution such as nicotine, coniine, and aniline. Another portion of the organs is extracted with alcohol, the alcoholic solution evaporated and the purified extract is treated with immiscible solvents such as ether, chloroform and benzol, which extract alkaloidal and some non-alkaloidal substances used as poisons.

After volatile solvents have been evaporated the residues are tested for alkaloids and non-alkaloid substances. In the latter class are the barbiturates, which are annually killing more and more people. They are frequently called "Goof" pills. Red Birds for seconal, Yellow Birds for Nembutal, Blue Birds for amytal, and Brown Birds for Delvinal. In New York City in 1947, eighty-seven people committed suicide by barbiturates, 58 were classed as accidental or undetermined, a total of 145 deaths. In Cook County the annual report of the Coroner's office for 1948, shows only 17 deaths by barbiturates. This number is far too low, probably due to a lack of post-mortems and routine chemical examinations. From the melting point of the purified crystals extracted from the organs, it is possible to determine the kind of barbiturate used. A micro-nitrogen determination of the crystals would help verify the identity.

In this brief discussion it would be impossible to go into the many details where the chemical examinations have solved the cause of death and helped clear up murder cases. In my forty years of experience as a Toxicologist I have seen the simple chemical tests changed to the less laborious optical and micromethods of chemical analysis, by the use of the spectrophotometer, spectrograph, polarizing microscope and the filtered ultra violet light. The "Black Light" excites fluorescent material, enables their detection in dilutions such as one part in 20,000,000. The toxicologist of the future will be an M.D., with a Ph.D. in physical chemistry.

THE LENGTH OF THE SECOND STAGE OF LABOR

A Review of the Literature

By MORTON JOSEPHSON, '49

The second stage of labor includes that interval between complete dilatation of the cervix and the expulsion of the infant.

In brief, the following course of events normally takes place during this interval. A few sharp pains, attended by a show of bright red blood usually indicates that the head is slipping through the cervix. The pains come every two or three minutes and are expulsive in character. The patient feels that she must force the infant out of her pelvis, and with the pain, having fixed the chest in inspiration, feet against the bed, she drives the head against the perineum by the powerful action of the abdominal muscles. Soon the labia part in the height of the pain and the perineum bulges. And then a large segment of the scalp becomes visible, receding after each pain, until a supreme effort forces the occiput out from behind the pubis, the forehead, face and chin following. In a few minutes, the renewed pains deliver the shoulder and finally, in a long, hard expulsive effort the trunk and lower extremities.

During this process, the fetus within, is going through a series of maneuvers. The first, descent, is the downward journey to the external world. It may begin in the first stage, or be entirely confined to the second stage. In the primipara engagement is frequently so deep at the onset of labor, the fetus having descended this far some weeks before labor begins, that the head is at the level of the ischial spines when labor begins so that the descent may be confined to the second stage. In multiparas, however, the descent begins with engagement of the head that usually occurs before dilatation of the cervix is complete.

Along with descent, as the head meets with the resistance offered by the cervix, the walls of the pelvis, and the pelvic floor, flexion results. In this moment the head rotates about its transverse axis in such a manner as to bring the chin into more intimate contact with the thorax.

The head at first descends in a transverse position. However, as the descent continues the head turns about its vertical axis in such a way that the occiput gradually moves from the position that it originally occupied toward, usually, the symphyis pubis. This enables the head to accommodate to expulsion through the pelvic outlet beneath the pubic symphysis.

When the head reaches the vulva, with the force of the uterus acting downward and the resistance of the pelvic floor acting upward, it is directed forward and somewhat upward causing extension, thus preventing impingement on the sacral floor and allowing the head to push forward unimpede with the occiput in direct contact with the inferior margin of the symphysis pubis. The scalp may now become apparent through the gradually dilating vulva, and more and more of the occiput appears, with the bregma, forehead, nose, mouth, and finally the chin successively passing over the anterior margin of the perineum. The head now rotates to the position it held before internal rotation occurred. Almost immediately after this external rotation, the anterior shoulder appears under the symphysis pubis, then the posterior shoulder, and finally the body of the child.[1]

According to Stander, the average length of these events takes about one hour in multiparas and from an hour and three quarters to two hours in primiparas.[2]

L. A. Calkins, who, with his co-workers, observed the second stage of labor critically, in 2,400 primiparas and 1,700 multiparas claims that good pains at frequent intervals may bring the descent of the head to the pelvic floor in 12 minutes in primiparas and in 6 minutes in multiparas. And even if the uterine contractions are weak, strong voluntary action of the abdominal muscles may produce descent in 10 to 15 minutes. Calkins claims that he considers the "push" of the parturient more effective than uterine contractions in hastening the spontaneous process of the second stage. He adds that another factor definitely related to the speed of descent is the size of the fetus, large fetuses averaging 6 minutes more for multiparas in this stage than small and medium sized ones. The delay in the case of the large fetus of the primipara is still greater. He also concluded that descent was completed more rapidly if internal rotation did

not occur until the presenting part was in the perineum.[3]

According to Herbert E. Schmitz and Janet E. Towne from their observations of 200 adolescent primiparas, the length of labor in all three stages is somewhat shorter for these primiparas than is considered normal for a primipara.[4]

Before leaving the discussion of the length of time in normal physiologic labor, it might be mentioned that today the second stage of labor is frequently shortened artificially by performing an episiotomy, sometimes followed by the use of prophylactic low forceps. The episiotomy is recommended by some obstetricians for all primiparas as it felt that the procedure prevents harmful stretching of the pelvic walls and floor and tends to prevent spontaneous perineal lacerations as well as facilitating delivery. By the same token, a multipara subjected to previous episiotomy with good repair is deserving of a similar procedure. Prophylactic low forceps are often applied after the episiotomy by some obstetricians because they believe that the artificial delivery of the head, before it is allowed to pound against the perineum for any length of time, prevents damage to the fetal head, saves wear and tear on the pelvic soft tissues and perineum and prevents exhaustive effort on the part of the parturient.

There are several factors that tend to alter the length of the second stage of labor, most of them affecting a prolongation of the process.

Cephalo-pelvic disproportion will prevent the head from engaging even after dilatation is complete and thus delay a normal descent. C. Nicholson and H. Allen in discussing this problem state that the condition is often the result of a defect in nutrition causing a deformed rachitic pelvis and that with general improvement in the dietary of children this danger to childbirth has been markedly lessened.[5] Hector R. MacLennan states as a word of caution, however, that the more minor rachitic deformities that occur today often lull the practitioner into a false sense of security. A pelvis may also be a congenitally poor passage. In pelvic disproportion it is the size rather than the shape that is important in childbirth and it is important, if possible, to recognize these discrepancies before labor ensues by the use of external and internal manual measurements as well as by roentgenology.[6]

William C. Ellen and William C. Mengert point out the fact that even in the presence of a normal pelvic inlet and outlet, there may be difficulties due to a mid-pelvic contraction and that recognition of this condition requires roentgenology. They add that it is strange that consideration of mid-pelvic contractions is often overlooked by obstetricians especially in view of the fact that published reports indicate that mid-pelvic measurements below the accepted normal of 10.5 cm. are common.[7]

With a normal pelvis, a large head, as in hydrocephalus, large babies as in diabetes of the mother, erythroblastosis, and precocious ossification of the skull bone of the fetus preventing proper molding, may delay the descent. Persistent transverse presentation in descent and at time of expulsion may delay or impede the second stage of labor. In these cases, the second stage is usually allowed to go two hours before forceps are applied.

N. Danforth states that in persistent occiput posterior position, ordinarily the second stage of labor should be terminated after two hours because the danger of either uterine rupture or the formation of a pathologic contraction. But as long as contractions are of less than normal intensity and as long as gradual descent is occurring and mother and child are in good condition, the case may be watched up to 5 hours, since the slow descent allows the head to better adapt itself to passage through the restricted diameter and by further molding enhances the ease of rotation.[8]

Uterine atonia may prolong the second stage. This may be due to idiopathic faulty physiology of the uterine muscles or paresis of the abdominal muscles, or perhaps, the fear of pain may cause the patient to be unwilling to use her voluntary muscles completely.[11] M. Speiser and G. Speck state that many multiparas approach the onset of labor poorly prepared to go through the ordeal of labor as a result of overwork, insufficient rest, anemia, and too frequent pregnancies, and these patients may have inertia uteri with resultant long labor.[9]

Another cause for delay is a functional contraction ring which may occur at the external or internal os or at any level of the lower uterine segment at the site of the physiologic ring or at any level of the upper uterine segment. These

may develop at any time from the beginning of labor to very late in the first stage or even during the second stage.[10]

Eclampsia may seriously impede the progress of the second stage of labor if the patient is permitted to deliver from below. Lack of cooperation on the part of the patient, her weakened state, the overwhelming sedation in the treatment and the convulsions are all factors involved in this type of dystocia.

Placenta praevia may also act as an impediment to labor for, as it occupies the lower portion of the uterine cavity, it interferes with the accommodation of the fetal head, and consequently abnormal presentations are unusually frequent.

An umbilical cord about the infant's neck or a short umbilical cord may also impede progress in the second stage.

Grantly Dick Read indicates how pain and the fear of it add to the duration of labor. He states that in labor, fear produces tension in the circular fibers of the uterus by stimulating the sympathetic nerve supply. Tension in these fibers opposes the action of the longitudinal muscle fibers supplied by the pelvic autonomic system. Increased tension in the lower uterine segment and cervix cause pain and pain intensifies fear until the exhausting agony of a relatively obstructed labor supervenes. The pain-fear-tension syndrome of labor may be controlled or avoided by correct interpretation of the pain stimuli by the parturient. And in the second stage, the downward thrust and retreat of the head before crowning must be explained since the phenomenon disappoints the woman if she doesn't understand it. Read also claims that the patient naturally relaxes between contractions in the second stage and wakes to labor at each pain with a will. As the head descends, onset of a dulling of consciousness between contractions may be observed. It is a kind of amnesia which a woman without an acquired fear of birth pains will normally inherit. He therefore recommends the development of a proper attitude toward labor pains as a means of shortening labor.[11]

W. D. Flath states that as far as demerol sedation is concerned, when demerol is given in adequate doses in the second stage of labor, labor progresses smoothly and often no anesthesia is required for delivery. Demerol does not depress uterine contractions in the first and second stages of labor, and occasionally, when given in the second stage, contractions become more effective, the pain threshhold is raised and delivery occurs sooner and more easily than would have been the case had no sedation been given.[12]

A report of the North Carolina Maternal Welfare Committee states that posterior pituitary extract will hasten the first and second stages of labor, but the danger of uterine rupture is ever present. The baby may incur physical damage due to the violent contractions, and a slowing of blood through the uterine placental sinuses may cause asphyxia of the fetus. Posterior pituitary extract is to be used in the second stage of labor only when the presenting head is in normal position and on the perineum so that only a few expulsive contractions of sufficient strength are necessary to complete delivery.[13]

Twins, by causing overdistention and thus inertia of the uterus, as sometimes also occurs in hydramnios, may cause a prolonged labor. The recent literature reports a case of locked twins that impeded the progress of labor[14] and another of siamese twins that had a similar effect.[15]

As far as breech deliveries are concerned, Schultze states that the serious disadvantages presented by a breech presentation concern themselves chiefly with the possible inability of the aftercoming and unmolded fetal head to enter the maternal pelvis.[16] Since, however, the occurrence of a breach presentation is favored by an obstacle which opposes the engagement of the vertex, such as contraction of the inlet of the pelvis, excessive size of the normal head or hydrocephalus, this may explain the frequent association of breech presentation and prolonged labor.

Uterine fibroids may also impede the progress of labor as well as extrauterine neoplasms. And, rarely, neoplasms of the fetus may have a similar effect. J. W. Boulard discusses a case of polycystic disease of the kidneys of a fetus causing an obstructing dystocia,[17] and J. J. Walls discusses a case in the recent literature in which the delivery ceased after the anterior and posterior shoulders were delivered due to a soft cystic mass that turned out to be a congenital sacral teratoma.[18]

A. C. Mallace discusses another infrequent cause of second stage dystocia, namely, ischiorectal abscess. He states that the prolonged labor is caused by the pain which prevents the patient

from bearing down and by the swelling which produces rigidity of the perineum.[19]

A final infrequent cause of dystocia reported recently by I. Siegel is that of a pelvic kidney obstructing a breech delivery.[20]

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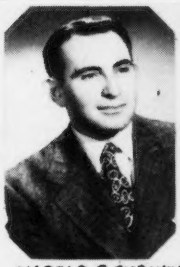

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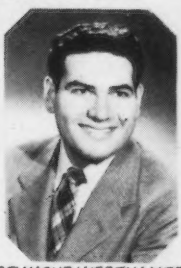

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By SEYMOUR KAPLAN, '49

It was hot and sticky, and beads of sweat tickled your neck; but you didn't remove your jacket or even loosen your tie. You were there. This was the day you had yearned for. You were in medical school at last, and this was your first class. You looked around you, and gazed at your classmates. They ranged from the extremely young, to the embarrassingly old. They came from all the corners of the land, and from all types of backgrounds. You were as heterogenous a group as one could find, and yet you were all here with the same thought. This was it. This was the tomorrow you waited for. This was now.

The lecture ended and you filed out nervously. Each fumbling with his hellos; each reassuring the other that he wasn't frightened. You bought your books and instruments, and the quarter was on.

How young you were, as was shown by the abuse and disrespect you heaved upon your cadaver to compensate for your terror. Remember those weeks of dissection and study? The countless explorations and adventures into the mystery of structure? The endless questions as, "Hey, doc! do we have to know the branches of the brachial plexus for tomorow's quiz? Hey, doc! what are these grey and white rami you're talking about, they all look pink to me?"

By the time you learned how to use your text, and learned to stare at your specimen's face, a new quarter started. You were then joined by a new group of students. You were all old hands by now and eagerly showed off your tremendous knowledge by offering to aid the fumbling neophytes to get started.

Chemistry was added to your course of study. Chemistry with its electrons, protons, dissociation constants and ionic equilibrium formulas.

"Hey, doc, what has the dissociation constant of NaOH got to do with medicine, huh!?"

Then came Bacteriology and Physiology. You were thankful you could hold on to three courses, but the school acknowledged that you were a brilliant student so what was two more courses. Remember the way the guinea pigs squealed when you injected tubercle bacilli into them? Remember how still they were when you saw the result?

In Physiology you were a surgeon first, and a physiologist second. "What ho, m'friend, give me a knife and tell me where to cut." But somehow, amid all the noise and confusion of the laboratory, and all the theories learned, came not only a basic knowledge of physiology, but what is even more important, a fundamental respect for living things, and an appreciation of the frailty and beauty of functioning tissue.

The quarter was over and you received a vacation. Remember how it was when you got home? How reluctant you were to show your texts to your friends? And how you answered, "Shucks, it's easy," when they inquired about the difficulty of studying medicine. Remember how your parents made you show them how you looked in your white uniform, and the blush of embarrassment when a neighbor called you "doctor"?

The vacation was over before it started and you were back at work. You looked around and noticed how the ranks had thinned. You felt safer now, a little surer that you'd make the grade after all. You could see yourself a graduate, and you began to worry. Your school's standing was in back of your mind now. As a freshman, graduation was years away, and you were positive that something would happen before you finished. As a sophomore you began to worry. The administration and the upper classes were selling their souls to aid the school, and now you entered the fight, with all you had, and you were hoping it was enough.

You were a pre-clinician. Pathology and Parasitology were added to the program. Pathology with its lessons of disease and response. This was it. This was what you were waiting for. You became so engrossed in your studies that you became yourself a living text of Pathology. The wart on your lip became a malignant melenoma. A cold became the onset of tuberculosis. And a muscle sprain made you certain that you had rheumatic fever. You were a pathologist now and you loved and feared it at the same time. In Parasitology you went purely and simply mad. Diphylobothrium latum, Taenia saginata and the Trypanosoma were created for the simple purpose of plaguing you.

Later came Clinical Pathology, Physical Diagnosis, Obstetrics, and finally Medicine. Stethe-

scopes began to pop up in your pockets, and a few of your hearty classmates even wore white jackets. Pediatrics, Pharmacology, enough work to swamp a genius, and yet you managed to pass them all. You were cleared by the Clinical Committee and became a Junior and a Clinician.

Oak Forest came first. Oak Forest—the city of the unwanted and the hopeless. Old and young, all with the same feeling of despair. You learned a great deal there. You learned how to talk to a patient, how to apply the knowledge you already had. Why, you even learned to relax a little. With Blue Island, Harvey, and Joliet nearby, your evenings were never lonely. You learned another lesson though, a lesson more important than all the rest. You learned something of the futility and limitations of medicine in the face of age and forlornness. You thanked God for your youth and your health.

That summer your spirits received a boost. Your school's endeavors began to reap dividends. The American Medical Association announced that the preclinical years were of an accreditable standard. There was hope at last.

Then came Cook County Hospital. The wards and wards of endless misery. At County you learned the technique of medicine and how to use the tools of your trade. Intravenous injections, plural taps, paracenteses, spinal taps, charting. You had your routine duties to perform and you had a part to play in the hospital day, and you loved it.

Later you went to Mount Sinai Hospital, and while it was the same it was also very different. You were new there, and yet you felt at home at once. This was where you belonged and you knew it.

A million lectures were thrown at you. There were a mere 21 courses and finals to pass and you'd be a senior. You caught your breath, kissed your girl friend goodbye, plunged in and crammed. Somehow, almost unbelievingly, you passed everything and were a senior.

You entered the clinics and were a "wiz" at once. Periarteritis Nodosa, Ayerza's disease; there was nothing you didn't diagnose. You knew ten times as much as the elementary juniors and at least five times as much as your instructors. However, you were young, and the young learn fast. You soon began to worry about your internship. Now was the time for the school to become approved, and yet it didn't. It was you up at bat now. You weren't on the sidelines anymore offering sympathy. You applied for an internship and prayed, "Let it come through, please, this month." And then, as if you really deserved it, the announcement was made, and you were approved.

At first there was just hysteria. The Chez Paree with your friends, the weekend parties, the dinner-dance in honor of Dr. Sheinin. The world was yours, imagine, a senior in an approved school. But as your senses began to return you began to ask, "Why?" You thought of all your friends who had gone before you. They were left out. They had struggled and sacrificed so much; they who had hoped and dreamed the same as you. It was largely on their merits that you were approved, not only your own. A deep humility entered and your attitude in the clinics changed. You became humble, you didn't know it all and you were scared.

It's late now. Comprehensives have come and gone. At first they seemed an impossible hurdle, but you've become used to that. You enter your task wearily, resigned and spiritless. You're tired and somewhat afraid. The results come out one by one. This one is clear, then another, oh, oh, some got caught on that one, are you among them? You passed them by like a weary soldier climbing obstacles. All have conquered to get home. The day in June comes closer and closer. There's the Senior Prom and the other socials. The rush of graduation preparations, and amidst them all there is still that feeling of humility and timidity. The day finally arrives. You receive your degree and wonder why. Surely, you don't know the first thing about medicine. You approach your family and friends who welcome you with tears and kisses. You smile at your parents and somehow your fear passes. You show them your degree and say, "Look, Mom, I'm a Doctor!"

The student body of The Chicago Medical School presented a portrait of the Dean as a permanent gift at a special ceremony held in the amphitheater of the school. Mr. Milton Miller of the Junior Class, introduced Mr. Bernie Halperin, Senior, who was chairman of the committee that commissioned Dr. Edward Giesbert, Associate Professor of Art at the University of Chicago, to paint the portrait of Dr. John J. Sheinin. In making the presentation of the painting, Mr. Halperin remarked that in years to come there will always be those who will be deeply grateful for all that the Dean has done unselfishly in behalf of the students.

Dr. Henry A. Smith, Secretary of the Board of Trustees, accepted the portrait for the school.

COMMENCEMENT 1949

The Thirty-Fifth Commencement of The Chicago Medical School was held on June 25, 1949, at the John B. Murphy Memorial Auditorium.

The Very Reverend Monsignor Aristo V. Simoni, Colonel, U. S Army, Retired, delivered the invocation and this was followed by an address by Clark George Kuebler, President of Ripon College, who stressed the values of our American heritage and our way of life which permits a freedom to choose our own destiny.

Dr. Edward Zucker received the first annual John J. Scheinin Meritorious Award, presented anonymously by a member of the staff of the Mount Sinai Hospital to a member of the Senior Class.

Mr. Lester N. Selig, Chairman of the Board of Trustees then conferred the Honorary Degree of Doctor of Laws to John Crippens Evans, distinguished presbyter in the Anglican Communion; leader in the cause of social justice; author of numerous publications on religious and educational matters; and untiring and loyal servant of The Chicago Medical School; and the Honorary Degree of Doctor of Science to John Jacobi Scheinin, distinguished teacher, scientist, author, and administrator in recognition of his outstanding achievement in medical education and yeoman service to The Chicago Medical School.

Dr. Scheinin then presented the thirty-nine candidates and Mr. Lester N. Selig conferred upon them the degree of Bachelor of Medicine.

School And Faculty Notes

SCHOOL NEWS

On June 11, 1949, in a simple ceremony held in Amphitheater A at 12:15 P.M., the student body of The Chicago Medical School presented the Library with a portrait of Dean John J. Sheinin, as a token of the high esteem which they hold for him and all that he has accomplished.

Dr. Henry A. Smith, Secretary of the Board of Trustees, accepted the painting from B. Halperin '49, spokesman for the entire student body.

FACULTY NEWS

We are pleased to learn that Dr. John R. Harger, Professor of Surgery, has recovered from injuries which he sustained in a recent automobile accident. . . . Dr. Piero P. Foa recently delivered a lecture, one of a series on Courtship and Marriage, at the Wabash Avenue YMCA.

Department of Physiological Chemistry

Dr. Ralph Goldfarb, of our Department of Chemistry, joined three other Chicago scientists who are studying radioisotope technique in research at the Oak Ridge Institute of Nuclear Studies.

Department of Anatomy

Dr. Edgar D. Congdon and Mr. Max R. Waller are making a census of ganglia for parasympathetic relay (probably of the ninth cranial nerve) to supply smooth muscle and glands of the oropharynx and larynogo-pharynx. The description of these structures has not appeared in the literature. Cross-section series of unilateral halves of the fetal pharynx are being used.

The sixty-second session of the American Association of Anatomists was held in Philadelphia on April 13-15. At the meeting, the following papers were presented by members of the Department of Anatomy:

Dr. Jacob M. Essenberg read a paper entitled, "Marked Abberations from Normal Coronary Patterns in the Human Fetal Heart, 22 Weeks of Age."

Dr. Harold S. Fish presented his paper on "Morphogenesis of Rat Connective Tissue During the First Ten Days of Life."

Dr. George Clark presented his thesis entitled, "Responses Elicited from the Anterior Limbic Region of Anaesthetized Dogs by Electrical Stimulations Through Fixed Electrodes." Assisting him in this project were K.L. Chow, C. Gillapsy and D. A. Klotz.

Dr. Leon H. Strong read and demonstrated his work on "Muscle Force Components in the Occlusive Mechanism of the Nasopharynx."

ALUMNI NEWS

Dr. Jerome S. Tobis, '43, announces the opening of his office in the Department of Physical Medicine and Rehabilitation of the Flower and Fifth Avenue Hospitals in New York City. . . . Dr. and Mrs. Arnold L. Berger, '43, announce the birth of their daughter, Andrea Susan, on April 21, 1949. . . . Dr. and Mrs. Stan Menachof, '45, announce the birth of their daughter, Mera Sue, on April 7, 1949. . . . Dr. Louise Tumarkin, '45, announces the opening of offices for the general practice of medicine at 7041 West Higgins Road, Chicago 31, Illinois. . . . Dr. Charles M. Biren, '46, announces the opening of his office for the practice of medicine, surgery, and obstetrics at 1985 Ocean Avenue, Brooklyn 30, New York. . . . Dr. Seymour Levine, '47, is now occupied with research on hyaluronic acid and hyaluronidase at the New York University Hospital.

PHI LAMBDA KAPPA NEWS

The annual Senior Dinner-Dance was held on May 21, at the Furniture Club of America, and a grand time was reported had by all of the more than two hundred persons who attended. The evening consisted of dinner, several short addresses, the presentation of keys to the seventeen senior members by Dr. Irving Siegel, a few songs by Bernard Kleppel, '49, and by David Safadi, '50, some comedy by the inimicable Messrs. Ludwig, '50, and Ostrove, '50, and dancing to the strains of Lucio Garcia and his N.B.C. orchestra.

FRESHMAN CLASS NEWS

Stuart Cohn will marry Miss Carole Jacobson on June 19, in Chicago.

Sheldon Loeb will wait slightly longer before making Miss Loretta Kirschenbaum of Cleveland his bride on June 26.

Henry Rosner, a more patient freshman, will

wait until September before making Miss Lillian Stein of New York, Mrs. H. Rosner.

Rowl Kowal was elected vice-president of the Freshman Class in a mid-semester election.

SOPHOMORE CLASS NEWS

Suzanne Allen '51 will marry Mr. Lee Widrow in New York City on June 25.

JUNIOR CLASS NEWS

The big news is the birth of a son, Jonathan, to David and Florence Safadi. The event took place at Mt. Sinai Hospital on March 22, 1949. Other members of the class who are soon to join the select circle of parents are the Larry Elegant's, the Milt Miller's, the Lenny Grayson's, and the Seymour Filman's. . . . Phil Oransky has been hit by a Bronx cupid! We had given him up for bachelorhood, but Miss Rhoda Herschander of the Bronx, N. Y. must have convinced him to try the sea of matrimony. . . . It has reached our ears that a matrimonial agent has been working on Dan Solomon during his last trip to Pittsburgh. His sister will soon be "Blessed Eventing." Congratulations Uncle Danny! . . . The entire Class extends its condolence to Jack Silberman upon the passing of his beloved father. . . . The Marvin Wertheim's celebrated their third wedding anniversary on May 30.

Larry Ravich has announced his engagement to Miss Sally Ann Miller of New York City. They will be married on June 26, 1949. Also to take their vows in the near future are Al Greenberg and Miss Rita Block of Chicago.

SENIOR CLASS NEWS

So many events have occurred recently in the Senior Class that one hardly knows where to begin. Hy Kaplan, who almost had everyone convinced that he was destined for many more years of bachelorhood, suddenly surprised one and all by marrying the former Miss Evelyn Cohn of Chicago, Ill. on March 26, 1949. That must have been a memorable day for Hy, especially since he had his Pathology Comprehensive on that same afternoon. . . . Jack Margolis was married to the former Miss Evelyn Pliskin of the Bronx, N. Y. on April 1, 1949, but poor Jack had to leave his young bride at home when he returned for the spring quarter since she is also a student — at Adelphi College in New York. . . . To add to the doings on April 1, Ray Firfer and Ann Platner announced their engagement. Ann is from Winnepeg, Canada, and is now an Anesthetist at Mt. Sinai Hospital here in Chicago. . . . Bill Schumer is another one of those who personally profited by Mt. Sinai Hospital's affiliation with The Chicago Medical School. He met Edith Westerfeld while a clerk there and it wasn't long before they decided to set the date for next November. Edith is an R.N. and was recently made Supervisor of the Fourth Floor. . . . Harry Surchin and Frances Rubin of Brooklyn, N. Y. (she is Bill Rubin's sister) are going to conclude a whirlwind romance at the altar on June 24, at the Sovereign Hotel in Chicago. They won't be able to start their honeymoon until the following day, however, 'cause Harry has to graduate. . . . Mort Josephson and Dorothy Katchkey of Sheboygan, Wisconsin (she is a student nurse at Mt. Sinai Hospital, Chicago) have announced their engagement. . . . Betty and Leon Yorburg have a new addition to their family, a baby daughter born at Norwegian-American Hospital on May 20 . . . Rut hand Abe Rosenstein celebrate their second wedding anniversary on June 29. . . . Seymour Kaplan and Natalie Solomon of Chicago will be married on June 26 in Evanston, Ill. They will then go to Oakton Manor in Wisconsin for several days before leaving for New York City where Seymour will start his internship at Harlem Hospital. . . . Al and Virginia Gasior-Russell are expecting an addition to their family "sometime this spring."

. . . . .

Graduating Seniors and Internship Addresses

Melville Alderman—
Hospital of St. Raphael, New Haven, Conn.
Sheldon Altman—
Harlem Hospital, New York, N. Y.
Harold Berson—
Coney Island Hospital, Brooklyn, N. Y.
Milton Bronstein—
Newark City Hospital, Newark, N. J.
Joseph Elgart—
Philadelphia General Hospital, Philadelphia, Pa.
Raymond Firfer—
Cook County Hospital, Chicago, Ill.
James Fletes—
Cook County Hospital, Chicago, Ill.
William Garrison—
Memorial Hospital, Springfield, Ill.
Alfred Gasior-Russell—
St. Francis Hospital, Evanston, Ill.

Harold Grushkin—
Harlem Hospital, New York, N. Y.
Bella Hearst—
(information not available)
Bernard Halperin—
Jersey City Medical Center, Jersey City, N. J.
Morton Josephson—
Harlem Hospital, New York, N. Y.
Jerome Justen—
St. Margaret Hospital, Hammond, Ind.
Hulbert Kanter—
Jersey City Medical Center, Jersey City, N. J.
Hyman Kaplan—
Cook County Hospital, Chicago, Ill.
Seymour Kaplan—
Harlem Hospital, New York, N. Y.
Sidney Kase—
San Bernardino County Hospital, San Bernardino, Calif.
Bernard Kleppel—
Mount Sinai Hospital, Chicago, Ill.
Marcia Levine—
St. Joseph's Hospital, Lancaster, Pa.
Samuel Libert—
Cook County Hospital, Chicago, Ill.
Jack Margolis—
Morrisania City Hospital, New York, N. Y.
Anthony Milea—
Harlem Hospital, New York, N. Y.
Marshall Persky—
Cedars of Lebanon Hospital, Los Angeles, Calif.
Louis Rosenbaum—
Harlem Hospital, New York, N. Y.
Abraham Rosenstein—
Charles S. Wilson Memorial Hospital, Johnson City, N. Y.
William Rubin—
Newark Beth Israel Hospital, Newark, N. J.
Joseph Saez—
Harlem Hospital, New York, N. Y.
Morton Schaffner—
Fordham Hospital, New York, N. Y.
William Schumer—
Mount Sinai Hospital, Chicago, Ill.
Leonard Singerman—
Mount Sinai Hospital, Chicago, Ill.
Nerissa Singh—
Mount Sinai Hospital, Chicago, Ill.
Walter Steiner—
St. Anthony Hospital, Chicago, Ill.
Harold Surchin—
Newark Beth Israel Hospital, Newark, N. J.
Arthur Upton—
Cook County Hospital, Chicago, Ill.
Eleanor Walters—
St. Mary's Mercy Hospital, Gary, Ind.
Seymour Werthamer—
Cumberland Hospital, Brooklyn, N. Y.
Leon Yorburg—
Bridgeport Hospital, Bridgeport, Conn.
Edward Zucker—
Harlem Hospital, New York, N. Y.

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(Continued from page 19)

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1949 Professional Schools Baseball Champions

Each Spring, under the sopsorship of the Professional Schools Young Men's Christian Association, has been the season for an inter-school softball tournament in which many of the neighboring schools participate.

This year, eight teams were entered, and winding up with an undefeated record were the Chicago Medical School Juniors. In all seven games our Juniors played they excelled, and it was through the fine spirit and sportsmanship exhibited that they set each succeeding team down to defeat. Under the very able managership of "Lippy" Hilton, the boys were able to bring home the "pennant," while the admirable pitching of captain "Rollo" Jaffee, as well as the sparkling and oftimes sensational fielding of Jack Silberman held the opposing teams to the barest minimum of runs. All in all, our Juniors scored a total of 99 runs, while amassing 108 hits from the opposing pitchers. The most impressive score was fashioned at the expense of our Freshmen, who were soundly drubbed to the tune of 23-5!

Other players on the Junior team include: Mel Ehrich, not in picture above, first base, Phil Oransky at second, John Guido at shortstop, Al Modert at the "hot corner," and Al Tucker who did a princely job at catching. In addition to Silberman, the outfield consisted of Abe Ludwig, Vince La-Rocca, Tony Fiorica, Ef Braverman, and Jerry Litt, with Fiorica substituting excellently as utility infielder.

A Short Bibliography on Health Insurance, Group Hospitalization Insurance and Medical Care

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Progress in Medicine

AUREOMYCIN

The most recent of the antibiotics to prove its clinical value is aureomycin. Derived from a strain of Streptomyces aureofaciens, the drug is supplied by Lederle Laboratories as a yellow crystalline hydrochloride salt in sealed vials containing 20 mg. It has been administered intramuscularly dissolved in isotonic sodium chloride solution and has also been used orally. Aureomycin possesses antibacterial activity against numerous Gram-positive and Gram-negative bacteria. It is bacteriostatic rather than bacteriocidal, except in high concentrations. Human serum has been found to have an inhibiting effect on the antibiotic's activity, and to obtain an antibacterial concentration in the presence of 50% serum about 50 times the concentration was necessary compared with that in broth.

The toxicity of aureomycin was determined in experiments on mice, rats, rabbits, and dogs. All mice survived a single intravenous injection of 50 mg. per kilogram, but only 14% survived 100 mg. per kilogram intravenously. Rats tolerated subcutaneous injections of 50 mg. per kilogram daily for eight days with only slight loss of weight. Three drops of both .25% and 1.0% solution of aureomycin borate in a rabbit's conjunctival sac caused no immediate or delayed reaction. Intravenous injection of 150 mg. per kilogram in dogs resulted in death in six hours — preceded by hyperpnea, anorexia, tremors, generalized paresis, somnolence, and hemoglobinuria. Dogs receiving 20 mg. per kilogram intramuscularly twice daily for nine days had anorexia and loss of weight. They also developed induration and necrosis at the site of injection, but there were no other untoward affects.

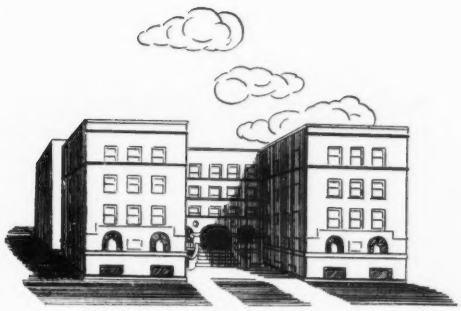
Aureomycin is rapidly eliminated in the urine and no significant blood concentration can be found in from one to three hours following the last injection.

In addition to the bacteriostatic properties, aureomycin has also exhibited virucidal properties, as was reported by Wright, et. al., in a series of 25 cases of lymphogranuloma venereum. The cases were divided into three groups clinically:

1. Those with buboes.
2. Those with lymphogranulomatous proctitis, with or without ulceration.
3. Those with benign cicatricial rectal strictures.

Group I consisted of eight patients, all Negro, aged 22 to 31. The lesion had been present in seven cases from two weeks to two months and in one case for two years. All had positive Frei tests and all patients exhibited unilateral nodes varying in size from a pea to an egg.

The Staff



Seven cases received 20 mg. of aureomycin intramuscularly daily, and two patients received in addition a single injection of 20 mg. of aureomycin into the bubo. All patients showed a marked reduction in the size of the gland after four days of therapy with aureomycin, "an event which in the experience over 24 years with several hundred cases with lymphogranuloma venereum has never occurred spontaneously within so short a time."

Group II consisted of three cases, two females and one male—ages 22, 25 and 27, respectively. There had been symptoms of rectal pain and discharge present for three weeks, one year, and two years. All patients were Frei positive.

Each patient received 20 mg. of aureomycin intramuscularly daily. The total doses given were 80, 200, and 220 mg. In two cases after four days the rectal pain and discharge ceased; in the third case rectal bleeding, which had also been present, disappeared after eight days. Proctoscopic examination at the end of the course of therapy in all three cases revealed no abnormalties of the rectal mucosa.

Group III consisted of fourteen cases, thirteen females and one male. The females ranged in age from 28 to 57; the male was 27 years old. Five of the patients had had previous colostomy for obstruction; one patient had a recto-vaginal fistula, and nine patients reported that the diameter of their stool was decreased. The durations of the lesions varied from two to twenty-six years with an average of 9.2 years, six cases had rectal pain, two had rectal bleeding, and seven patients had a rectal discharge. All were Frei positive.

These patients received 10 to 20 mg. of aureomycin intramuscularly daily, the total dose ranging from 160 mg. to 1 gm. After receiving the course of therapy six patients showed stools twice their previous diameter; two cases showed increase in diameter of the stool; rectal bleeding ceased; discharge stopped in six patients; and in the six cases with rectal pain, it disappeared during therapy.

Ross, et. al., reported on the use of aureomycin in the therapy of thirteen cases of Rocky Mountain Spotted Fever during the summer of 1948. Their study indicated that clinical results were obtained with the daily use of 30 to 60 mg. of aureomycin per kilogram of body weight, given orally. The intravenous and intramuscular routes of administration were not used in these cases. There were no toxic manifestations reported, although four cases experienced nausea and vomiting during the first two days of therapy. The rash disappeared in three to five days when therapy was instituted early. In two cases where use of the drug was begun on the eighth day of the disease, the rash was slow to fade and was still present after one week. Most patients on admission to the hospital were toxic, anorexic, and lethargic. Within 24 to 48 hours after starting aureomycin the patients were active, alert, and showed a return of appetite and interest in their surroundings.

O'Leary, et. al., reported on another very vital and very interesting aspect of the oral use of aureomycin — that is, its effect on Treponema pallidum in the human being. The drug was used in dosages of the order of the 500 to 750 mg. every four hours. This was dispensed in the form of capsules containing 250 mg. per capsule. Vitamin supplements were used because of the marked suppression of the intestinal bacterial flora and the appetite which resulted after administration of large doses of aureomycin. Two cases were reported.

The first patient had a superficial ulcer (4 cm. in diameter) on the

scrotum. Material obtained from the ulcer revealed Treponema pallidum on dark field examination. The patient was hospitalized and received from 400 to 750 mg. of aureomycin every four hours for a total of 44.2 Gm. A febrile Herxheimer reaction was noted 24 hours after therapy was begun. At 60 hours after the onset of therapy the dark field examination was negative and it remained so. The Kline, Kahn, Hinton, and Kolmer tests all remained positive but the titre decreased from 256 Kahn units to 32 Kahn units. There was no emesis and no avitaminosis; the only untoward reactions being nausea and anorexia.

The second patient presented a penile lesion (an ulcer 1 cm. in diameter) of one week's duration. The dark field examination for Treponema pallidum was positive. The Kahn, Kline, and Kolmer tests were negative; the Hinton was positive. The patient was given 750 mg. of aureomycin every four hours, orally, for a total of 67.5 Gm. Sixteen hours after the onset of therapy the dark field became and remained negative. The primary lesion healed completely. The only untoward effects were nausea, anorexia, one instance of vomiting, and the tongue was smooth and red indicating an early vitamin B deficiency.

Thus, with the advent of aureomycin, medical science has added a new and potent weapon to the struggle of mankind against the bacterial and viral diseases which heretofore have been so rampant.

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RECENT ADVANCES IN THE THERAPY OF NEOPLASTIC DISEASES

THE search for chemicals to control cancer has followed a course much like the one pursued over the years by those who wished to transmute the elements. Every age has had its alchemists and every one its cancer remedy. The confidence of the credulous has been so repeatedly betrayed that it has recoiled from the repeated failures. A long series of bitter disappointments has led to a conditioned reflex: the view that all who search for new means of cancer control are idyllic dreamers, blissfully unaware of the impossibility of ever attaining their goal. In spite of alchemists and sceptics, however, transmutation is now a fact. Similarly, notwithstanding past failures, mistakes, and confusion, progress in cancer therapy now is being made. It is as yet enormously ill-defined, vague; stirring and struggling of an amorphous mass of scientific facts, yet its tendency to take form and to move toward a definite goal is now both apparent and real. (1)

This review will be limited to the field of chemotherapy. The ideal agent which will selectively affect the neoplastic cells without seriously impairing the normal ones has not been found as yet, but some substances or groups of substances are available and seem to offer some hope. Among these are: 1) the nitrogen mustards, 2) urethane, and 3) stilbamidine.

1. The Nitrogen Mustards

The story of Nitrogen Mustards started during World War I, when Mustard Gas or Yellow Cross Gas was used on a relatively large scale and was classified as a vesicant. It was shown at that time that dichlorethylsulphide or mustard gas produces skin lesions very similar to those produced by X-rays and that, like X-rays, it strongly affects the bone marrow and the lymphatic system. During the interval between the two World Wars, and especially during World War II, a

large number of mustard gas analogues were prepared by substituting nitrogen for the sulfur of mustard gas and attaching to it various alkyl radicals. The nitrogen mustards are selective poisons of rapidly growing and dividing cells, they stop gametogenesis in the drosophila, produce mutations in the drosophila and in the Neurospora, abolish mitosis in growing embryos and, in the adult animal, inhibit the proliferation of actively proliferating tissues like the bone marrow, the lymphatic tissue, the intestinal mucosa, etc.

The nitrogen mustards are quarternary ammonium derivatives; they are very unstable and, when dissolved in water or tissue fluids, rapidly form cyclic onium cations which react with a variety of biologically important chemical groupings (amino-, carboxyl-, sulfhydryl-, etc.) altering the chemical and immunological properties of body proteins, and inactivating essential enzymes (2).

The strong cytotoxic action of the nitrogen mustards and the similarity of their action with that of the X-rays prompted several investigators to use these products in the therapy of experimental malignant tumors. The first results were encouraging as large masses of sarcoma 180 of mice could be made to dissolve after relatively few injections. The regression of the tumor was, however, not permanent and the mice eventtually succumbed. There followed a long period during which the toxicity of the N mustards was studied and finally the first therapeutic attempt was made on six terminal cases of malignant disease in man (2). All six patients died, but a rapid dissolution of large lymphosarcomata was obtained in two cases. Since this first experiment several hundred cases of malignant disease have been treated with the two most effective and least toxic of the nitrogen mustards (methyl-bis-beta-chloroethyl-amine hydrochloride and methyl-tris-beta-chloroethyl-amine hydrochloride). C. P. Rhoads (1) has recently summarized the present status of this therapeutic experiment. His conclusions are as follows:

1. The methyl-bis compounds are preferable because they have less tendency to thrombose the vein at the site of injection.

2. The recommended dosage is 0.1 mg per kg intravenously on four successive days.

3. The treatment is handicapped by toxic effects, such as: severe local inflammatory reaction if the material escapes out of the vein at the site of injection; nausea and vomiting occurring 1 to 8 hours after the injection and continuing for 3 to 24 hours (sometimes associated with anorexia, weight-loss, weakness and headache); and damage to the bone marrow with leukopenia, anemia and thrombocytopenia.

4. The nitrogen mustards do not perform cures for the diseases which have been studied up to the present; in large doses they are injurious to many types of tissues especially the rapidly growing ones, either normal or neoplastic; their toxicity for the bone marrow determines the dose which can be safely given a patient; tumor regression brought about by these compounds is temporary and rarely lasts more than several months.

5. Hodgkin's Disease may respond to nitrogen mustard therapy with remissions lasting 4 to 8 months, especially in early and slowly progressing cases; in the rapidly growing lymphosarcoma the results are usually insignificant, but the less malignant types may respond to therapy with regression of the tumor masses and reduction of the white cell count. The compounds seem to be of little or no value in the treatment of acute leukemias, although they are sometimes useful in the chronic forms.

It may be added that the best therapeutic results have been obtained in cases of polycythemia vera and in Hodgkin's Disease. In the latter disease symptomatic relief may be prompt and there may be dramatic improvement of systemic symptoms, such as fever, anorexia and pruritus. Nitrogen mustards are particularly useful in cases of Hodgkin's Disease which have become resistant to X-rays.

It may be concluded that Nitrogen Mustards are not a cure for malignant diseases, but they are a useful therapeutic adjunct. It is possible that further study of their mechanism of action may lead to further advances in the chemotherapy of cancer and in our understanding of the disease.

2. Urethane

The discovery of growth effects of certain urethanes on experimental cancers led to the trial of urethane in patients with inoperable cancer, principally, advanced carcinoma of the breast. Although the results were most disap-

pointing, it was noted that urethane produced a fall in the leukocyte count in some cases. The drug was therefore tried in cases of leukemia and allied disorders. Peterson and collaborators reported that urethane produces remarkable changes in leukemia: lowering of the total leukocyte count to normal, more nearly normal pattern of the differential count, reduction in size of the spleen and lymph nodes, and rise in the hemoglobin level. The authors conclude, however, that "there is no indication that permanent benefit may result from the use of urethane in either myeloid or lymphatic leukemia; for relapses take place and immature cells may reappear in the blood. The cases are too recent to enable any statement to be made about the effect of treatment on length of life. It can, however, be said that the palliative effect is in many cases very great." The treatment is not without danger as it has been reported that urethane has a tendency to produce pulmonary edema and at least two cases of death from it have been reported.

3. Stilbamidine

Another chemical agent which has shown promise in cancer therapy is stilbamidine. This drug was first used in the treatment of kala-azar and in experimental trypanosomiasis. Because kala-azar is accompanied by an increase in serum globulin, Snapper (4) made a trial of the drug in multiple myeloma which is also associated with high serum globulin. In 14 patients with severe forms of the disease, Snapper found relief of the severe bone pain, in 9 of the 14 cases the disease was arrested and 2 complete invalids were able to resume their normal activities. This improvement had lasted 16 months at the time of publication of Snapper's paper. The clinical improvement was associated with some recalcification of the bone lesions. Although the disease was arrested by stilbamidine therapy, it was not cured, since myeloma cells were still found in the bone marrow.

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HYALURONIDASE

In 1928, Duran-Reynals discovered that extracts of rodent testes greatly increased the infectivity of vaccine virus upon intradermal injection in rabbits. The effect appeared to be due to a faster "spreading" of the vaccine from the site of injection. It was soon found that the spreading effect of testes extract was not limited to vaccine, but that it affected also dyes, india ink, rabies virus, hemoglobin, diphtheria toxin and other substances. It was also found that a "spreading factor" was present in a variety of biological material such as bacteria, snake venom, certain tumors, placental and embryonic tissues, leeches and others. The most important of the group of substances known as spreading factors is hyaluronidase, an enzyme widely distributed in nature.

This enzyme has the property of hydrolizing hyaluronic acid, one of the polysaccharides constituting the ground substance of connective tissue and a constituent of the vitreous humor, the gelatinous material of the umbilical cord, as well as of some microorganisms. Hyaluronic acid has also been found in subcutaneous tissue of cases of myxedema, in the pleural fluid of patients with pleural endothelioma, in synovial fluid, fowl leucosis, virus tumors, streptococcus hemolyticus, etc. It is prepared from extracts of bull testes. The chemical basis of the spreading action appears to be the depolymerization and hydrolysis of hyaluronic acid, with "dissolution" of the mucoid substance of the connective tissue. In this manner the penetration of any material into the body tissue is facilitated. Hyaluronidase has been invoked to explain the invasiveness of certain bacteria, the relative low cohesiveness of cancerous cells, increased sedimentation rate in certain diseases, the dispersion of the corona cells of the ova which makes them susceptible to penetration by the spermatozoa.

The spreading action of hyaluronidase has been put to practical use. It has been used in conjunction with local anesthetic agents in surgery and dentistry, it has been added to fluids injected by hypodermoclysis to facilitate their absorption, in the treatment of human infertility either by injection to the male or by application to the cervical canal in the female, to facilitate the absorption of iodine dyes in urography of infants when the dye is administered by clysis.

The fact that iodides activate hyaluronidase has been invoked to explain the well known action of iodides in resolving certain types of inflammatory tissues.

VITAMIN B_{12}

This vitamin has recently been isolated as red crystals from purified liver extract and is believed to be identical with the erythrocyte-maturing factor. It is a cobalt and phosphorus complex containing one atom of cobalt and three atoms of phosphorus per molecule. A ton of fresh liver yields only about 250 milligrams of vitamin B_{12}, but, on the other hand, the material is one of the most potent known to medicine as a maximal reticulocyte response and a near maximal erythrocyte response can be obtained in pernicious anemia with a single injection of 5 to 10 micrograms. The vitamin is active in other forms of macrocytic anemia such as nutritional macrocytic anemia, tropical and non-tropical sprue and others. Macrocytic anemia of pregnancy and of infancy and childhood sometimes fail to respond to vitamin B_{12} and responds to folic acid or to combined therapy. At the present time the primary indication for vitamin B_{12} is pernicious anemia as the vitamin seems not only to give an excellent hematologic response, but to correct the changes in the bone marrow and improve the neurologic symptoms. In its effectiveness against the subacute combined degeneration B_{12} differs from Folic Acid which is inactive and, according to some authors, may accelerate the progress of the nervous lesions. The similarity of action between liver extracts and vitamin B_{12}, as well as the fact that potent liver preparations contain significant amounts of the vitamin suggests that vitamin B_{12} might be identical with the erythrocyte maturing principle of the liver. Although the clinical reports have been limited in number and the time elapsed from the beginning of B_{12} therapy necessarily short, the vitamin has proved itself a very valuable therapeutic aid, especially in cases of allergic reaction to liver extracts. It is possible that vitamin B_{12} alone or in combination with liver extract may prove to be the treatment of choice for pernicious anemia. For the present, however, the clinician should continue to use liver extract or be prepared to switch back to it if the patient does not make satisfactory progress.

THE PHYSICIAN'S WIFE

Did you ever wonder as the years trod by,
  As he brought new souls, saw others die,
What kept his fire of strength alive;
  What made him never ending strive
To help another, sick and pained,
  To spirit a lonely, aching and maimed.

Did you ever wonder how he did it all,
  How he managed to visit the great and the small,
To offer his skill and wisdom so wide,
  His comfort, and courage, his all he applied.

Did you ever wonder if fun he knew,
  Slaving so busily the whole year through,
Or whether he'd ever relax for a day,
  Whether he'd ever find time to be gay.

And wonder you might as you look at this man,
  Doing a job beyond reason and plan,
But wonder you needn't if you were to know,
  The spirit that pressed and never let go.

For that very great force, though hidden from sight,
  The force that created the spirit and fight,
Was just a small hand, a nod or a praise,
  A touch of endearment, an amorous gaze.
From the woman he loved, the all of his life.
  The greatness of him, THE PHYSICIAN'S WIFE.

—*RUTH ROSENSTEIN*

Book Review

HANDBOOK OF MATERIA MEDICA, TOXICOLOGY, AND PHARMACOLOGY by F. R. Davison. Fourth Edition, 1949; C. V. Mosby Company; 730 pages; $8.50.

This book, as its title suggests, presents a very comprehensive and compact survey of materia medica, toxicology, and pharmacology, and the information is made readily available by the use of a very complete 25-page index.

The author must have spent many exhausting hours in arranging and pruning his material for there are no superfluous words or sentences. When you want information about a particular drug you will find it, concisely written, so that many pages of discussion need not be delved into to secure the particular passage which is an answer to your query. The author devotes two chapters to materia medica and prescription writing, and reading these pages would greatly benefit the student who will find many things which are new, and the practitioner who will re-discover many things which are old.

There are also many well written chapters on toxicology, a classification of drugs, drugs acting on the skin and mucous membranes, antiseptics and disinfectants, drugs acting on the central nervous system and drugs acting on the peripheral nervous system.

In addition, Dr. Davison devotes many pages to a thorough understanding of the more recent developments in drug therapy in his chapters on cardiovascular drugs, oxytocics, diuretics and biologicals, including vitamins, serums, vaccines, blood, blood derivatives, blood substitutes, hematinics, and hormones.

Finally, in closing, Dr. Davison discusses briefly the antihistaminics, protein hydrolysates, gold therapy, snake venom, helium as used in severe asthma, and fluorides as applicable to dental caries.

The book is very well written and illustrated and would be an asset to any reader — be he student practicing physician, or professor.

* * * * *

FUNDAMENTALS OF INTERNAL MEDICINE by W. M. Yater. Third Edition, 1949; Appleton-Century-Crofts, Inc.; 1451 pages; $12.00.

This book has always been a favorite since it was copyrighted, in 1938, because of its clarity, its completeness without being verbose, and its excellent illustrations which make the passages a bit more meaningful and a bit more striking to its readers. Dr. Yater and the others who collaborated with him have outdone themselves in this new edition. The book shows painstaking organization and the material printed has the freshness of a recent journal on experimental medicine while discussing the diagnosis, differential diagnosis, and treatment of the traditional pathologic entities, as well as those of more recent repute.

The text is divided according to subject matter into twenty-six chapters, each of which has been written by an outstanding authority in that particular field. Included are such broad topics as diseases of the heart, blood vessels, kidneys, blood and blood forming organs, respiratory system, digestive system, locomotor system, endocrine glands, reticulo-endothelial system, metabolism, allergy, intoxications, vitamin deficiencies, malnutrition, infectious diseases, nervous and mental diseases, and diseases of the skin, ear, and eye. There are also discussions relating to dietetics, chemotherapy and therapy with antibiotics, symptomatic treatment, inhalation therapy, and useful clinical values; but placed at the very end is a chapter written by Dr. Yater and entitled, "The Physician Himself," which I found extremely interesting reading. Dr. Yater offers many helpful suggestions which could be read and digested profitably by any medical student or interne who is contemplating going into private practice.

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HOW TO BECOME A DOCTOR. By G. R. Moon; 131 pages; illustrated. The Blakiston Co. 1949. $2.00.

This book was designed primarily for pre-medical students in order to give them some insight on how to apply to a medical school and the common problems they will have to face after they have embarked on their medical education. It is a well written book by a man who has had

20 years of experience with medical students and schools, and Mr. Moon goes into a detailed discussion and analysis of the problems of finances, housing, outside employment, internships, and residencies. There is also an analysis of all of the medical colleges in the United States and Canada.

For those who are interested in following one of the other professions, Mr. Moon has also included the fields of dentistry, veterinary medicine, pharmacy, optometry, chiropody, occupational therapy, hospital administration, medical illustration, and science.

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